Single-Cell Transcriptomic Profiling of Esophageal Squamous Cell Carcinoma: Dynamic Tumorigenic Trajectories and Transitional Signatures of Oncogenic Evolution

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Using single-cell transcriptomic profiling, investigated the functional and expression alterations in esophageal epithelial cells migrating from normal to ESCC. The cells were taken from a mouse model of ESCC caused by 4NQO. A total of 1756 mouse esophagus epithelial cells were studied, which were divided into six subtypes. A single-cell diffusion map of these cells revealed distinct variances during their passage through all clinical stages, as well as significant modifications as cells progressed towards hyperplasia and aggressive cancer. The distribution of marker genes such ALDH3A1, ATF3, S100a8, ITGA6, and MMP14 varied depending on the stage of the disease. The sudden overexpression of S100a8 in cells during the hyperplasia stage suggested that the esophageal tissues underwent a major immunological shift.

T cell status during ESCC start was also investigated, with CD8+ T cells and CD4+ T cells separated into seven clusters each. During 4NQO-induced ESCC carcinogenesis, T cells were found to have diminished anti-tumor activities and increased inflammatory responses. The interactions between inflammatory immune cells and malignant epithelial cells increased, according to interaction analyses. These exposures imply that the inflammatory microenvironment may promote esophageal epithelial cell malignancy. Finally, we confirmed that similar changes occur in human ESCC tissues by validating the gene expression profiles in human ESCC specimens.

The single-cell transcriptomic profiling of distinct cell types at various pathogenic phases during 4NQO-induced ESCC carcinogenesis is described in this short communication.

We created an atlas of the malignant transformation of epithelial cells exposed to 4NQO based on these exposures. At different stages of carcinogenesis, the transition landscapes of immune cells and fibroblasts in tissue microenvironments were also depicted. This exposure will aid in understanding of the onset and progression of ESCC, as well as establish the groundwork for the development of molecular markers for early detection and precise treatment options for ESCC; nevertheless, there are numerous limitations to this short communication. To begin, single-cell transcriptomic profiling was

removing the complete esophagus epithelial layer. Third, the fates of epithelial cells and esophageal microenvironment transitions generated by 4NQO were discovered; however, the underlying molecular pathways were not investigated. Despite these constraints, the transition status and transcriptomic changes of several cell types in the esophagus during the development of ESCC were identified.